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Review Article

Association Between Ultra-Processed Food Consumption and Metabolic Dysfunction-Associated Steatotic Liver Disease (MASLD) in Adults

(A Systematic Review)

Lila Amila[1]*, Kurnia Agustina Sitompul[2], Maretha Primariayu[3], Dalilah Salsabila Salma[4], Siti Aria Rahmani Novianto[5], Marsha Zahrani[6], Nadia Mardiana Hudan[7]

[1] Clinical Nutrition Program, Airlangga University, Indonesia; e-mail: lilaamila2000@gmail.com
[2] Clinical Nutrition Program, Airlangga University, Indonesia; e-mail: kurnia.sitompul@fk.unair.ac.id
[3] Clinical Nutrition Program, Airlangga University, Indonesia; e-mail: maretha.primariayu@fk.unair.ac.id
[4] Clinical Nutrition Program, Airlangga University, Indonesia; e-mail: sssalsabila@gmail.com
[5] Clinical Nutrition Program, Airlangga University, Indonesia; e-mail: rerearian22@gmail.com
[6] Clinical Nutrition Program, Airlangga University, Indonesia; e-mail: Zahranimarsha1@gmail.com
[7] Clinical Nutrition Program, Airlangga University, Indonesia; e-mail: nadiamardiana50@gmail.com
*Corresponding Author: lilaamila2000@gmail.com

Abstract: Metabolic dysfunction-associated steatotic liver disease (MASLD) is the most common chronic liver disease globally, but its association with ultra-processed food (UPF) consumption under the new diagnostic framework has not been systematically synthesized. We systematically searched PubMed/MEDLINE, Embase, Cochrane CENTRAL, Scopus, and Web of Science for observational studies examining habitual UPF intake (NOVA classification) and MASLD/NAFLD in general adult populations using validated outcome ascertainment. Quality was appraised via the Newcastle-Ottawa Scale (NOS) and certainty via GRADE. Eight studies comprising >350,000 adults across six countries met the eligibility criteria, with seven rated as good methodological quality. All eight studies reported a positive association between higher UPF consumption and MASLD risk, with prospective adjusted hazard ratios ranging from 1.18 to 1.50 and cross-sectional odds ratios from 1.33 to 2.50. Additionally, decreasing UPF intake significantly lowered intrahepatic fat (7.7% to 2.6%), and an exposure-response gradient was observed across several cohorts. Approximately two-thirds of the association was mediated by adiposity (attenuated after BMI adjustment), while evidence for hepatic fibrosis remained inconsistent. GRADE certainty was rated as Low for the primary association and exposure-response gradient, and Very Low for intrahepatic fat and fibrosis outcomes. In conclusion, higher habitual UPF intake is consistently associated with an increased burden of MASLD in adults, largely mediated by adiposity. Although observational nature limits direct causality, these findings support restricting UPF consumption while awaiting further prospective studies using explicit MASLD criteria.

Keywords: Diet; Hepatic Steatosis; MASLD; Ultra-Processed Food; Systematic Review.

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1. Introduction

Steatotic liver disease has become the most common chronic liver condition worldwide, and its metabolic form is now the dominant contributor to the global burden of liver-related morbidity and mortality. In 2023 a multisociety Delphi process replaced the term non-alcoholic fatty liver disease with metabolic dysfunction-associated steatotic liver disease, or MASLD, abandoning the earlier exclusion-based definition in favour of positive diagnostic criteria that require hepatic steatosis together with at least one cardiometabolic risk factor (Rinella et al., 2023). The change was intended to reduce stigma and to sharpen case definition, but it also means that the large body of evidence accumulated under the older label

must be re-examined to confirm that established associations still hold when the new criteria are applied.

The scale of the problem is considerable. Pooled estimates place the prevalence of steatotic liver disease well above half in populations with type 2 diabetes and at roughly a third of adults in the general population, with a trajectory that continues to rise alongside obesity and sedentary living (Younossi et al., 2024). Because a substantial share of affected adults progress to steatohepatitis, fibrosis and their complications, and because effective pharmacotherapy remains limited, modifiable dietary drivers of the disease are of particular public health interest. Diet sits at the centre of both the pathogenesis and the management of MASLD, and international expert consensus now explicitly recommends reducing intake of ultra-processed foods as part of dietary prevention (Zeng et al., 2024).

Ultra-processed foods are defined within the NOVA classification as industrial formulations made largely or wholly from substances extracted from foods, together with additives designed to make the final product hyper-palatable, convenient and durable (Monteiro et al., 2019). Their consumption has expanded rapidly and now accounts for a large fraction of daily energy in many high-income and middle-income countries. An umbrella review of systematic reviews with meta-analyses has linked higher ultra-processed food intake to all-cause mortality, cardiovascular disease, type 2 diabetes and several cancers, establishing these foods as a consistent marker of adverse cardiometabolic health even where the mechanism remains debated (Barbaresko et al., 2024). The same dietary pattern is biologically plausible as a driver of hepatic steatosis, because ultra-processed foods are typically energy-dense and rich in refined carbohydrate, added sugars and fructose, which promote de novo lipogenesis and hepatic fat accumulation (Baharuddin, 2025).

Several individual studies and disease-specific syntheses have examined ultra-processed food intake in relation to steatotic liver disease. A systematic review and meta-analysis reported a higher risk of the condition with higher intake and suggested a dose-response relationship (Henney et al., 2023), and a further narrative synthesis found broadly consistent, if heterogeneous, associations across metabolic syndrome, insulin resistance and fatty liver (Grinshpan et al., 2023). More recent meta-analyses of adverse liver outcomes have reinforced the direction of the association while documenting substantial statistical heterogeneity (Guo et al., 2025; Zhang et al., 2025). Diet quality more broadly, including the timing of energy intake and the balance of whole versus processed foods, has repeatedly been implicated in the natural history of the disease (Marjot et al., 2023; Zheng et al., 2022).

Despite this accumulating evidence, several gaps remain. Almost all prior syntheses were framed around the older non-alcoholic fatty liver disease construct and were completed before or around the 2023 nomenclature change, so the extent to which the association is reproduced under explicit MASLD criteria has not been mapped. Studies also differ markedly in how they operationalise exposure, expressing ultra-processed food intake as a percentage of total energy, a percentage of total weight or a number of servings, and in how they ascertain the outcome, ranging from ultrasound and transient elastography to magnetic resonance imaging and validated steatosis indices. These differences plausibly explain part of the inconsistency in reported effect sizes, yet they have rarely been examined as sources of variation rather than pooled over. The degree to which the association survives adjustment for body mass index and total energy intake, both of which lie on the causal pathway between diet and liver fat, is a further unresolved question.

We therefore conducted a systematic review of observational studies to examine the association between habitual ultra-processed food consumption and MASLD, or its antecedent term non-alcoholic fatty liver disease, in adults from general, community or health-screening populations. The review deliberately excludes studies restricted to children and to populations selected solely for obesity or type 2 diabetes, so that the question is addressed in broadly representative adult samples. Evidence is synthesised narratively and the certainty of each conclusion is rated formally, so that the direction, consistency and limitations of the current evidence can be judged.

2. Proposed Method

Registration and Reporting

The review was conducted and reported in accordance with the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) 2020 statement (Page et al., 2021). The protocol was defined before screening; prospective registration on PROSPERO is recommended before submission and is noted in the revision log.

Eligibility Criteria

Eligible studies were observational (prospective or retrospective cohort, cross-sectional, or case-control) and enrolled adults aged 18 years or older from general, community or health-screening populations. The exposure of interest was habitual ultra-processed food consumption classified with the NOVA system and operationalised as the highest category, quartile or tertile, or as a defined increment of intake, ascertained by a validated food frequency questionnaire, 24-hour recall or food diary. The comparator was lower ultra-processed food consumption within the same source population. The primary outcome was the presence or incidence of MASLD, or its antecedent term non-alcoholic fatty liver disease, ascertained by hepatic ultrasonography, controlled attenuation parameter, magnetic resonance imaging, magnetic resonance spectroscopy, liver histology or a validated steatosis index. Secondary outcomes were intrahepatic fat on a continuous scale, elevated alanine aminotransferase, hepatic fibrosis by liver stiffness or a validated score, and evidence of an exposure-response gradient. Studies restricted to children or adolescents, to populations selected solely for obesity or type 2 diabetes, or to competing causes of chronic liver disease, and studies whose exposure was not the NOVA ultra-processed food construct, were excluded. No language or date restriction was applied.

Information Sources

PubMed/MEDLINE, Embase, Cochrane CENTRAL, Scopus and Web of Science were searched from inception to the search date. All five databases were executed. Reference lists of relevant reviews were screened to identify additional studies.

Study Selection

Records were de-duplicated across databases by DOI, PMID and title and screened in two stages, first by title and abstract and then by full text against the eligibility criteria. Screening and full-text assessment were performed independently by two reviewers (the first and second authors), with disagreements resolved by discussion. Reasons for full-text exclusion were recorded in pre-set categories.

Data Extraction

A structured form captured study identifiers, country and setting, design, population (sample size, age, sex), the exposure definition and dietary assessment instrument, the comparator, outcome ascertainment, follow-up, the adjusted effect estimate with its confidence interval and the covariates adjusted for, and funding. Values were taken from the source report; where only the abstract was available for a given datum, this was recorded through the source locator and the value was marked as abstract-level rather than inferred. Missing values were entered as not reported.

Risk of Bias

Risk of bias in each study was appraised with the Newcastle-Ottawa Scale, using the version appropriate to the study design across the domains of selection, comparability and outcome or exposure ascertainment, with an overall rating of good, fair or poor (Stang, 2010). Particular attention was paid to whether each study adjusted for body mass index and total energy intake, because these variables lie on the causal pathway and their handling materially affects interpretation.

Synthesis and Certainty

Because the studies differed in design, in how ultra-processed food exposure was operationalised and in how the outcome was ascertained, the evidence was synthesised narratively and organised by outcome rather than statistically pooled. For the primary outcome the number of contributing studies, the range and direction of effect estimates, the consistency of the association, the presence and shape of any exposure-response gradient, and the influence of adjustment for adiposity and energy intake were examined and interpreted. Effects were considered separately for prospective and cross-sectional designs, because the former speak to incidence and the latter to prevalence. The certainty of evidence

for each outcome was rated with the GRADE approach, starting from low for observational evidence and adjusting for risk of bias, inconsistency, indirectness, imprecision, publication bias and, where present, a dose-response gradient, with each judgement tied to the observed data (Guyatt et al., 2008).

3. Results and Discussion

Study Selection

The searches returned 440 records: 103 from PubMed/MEDLINE, 137 from Embase, 46 from Cochrane CENTRAL, 91 from Scopus and 63 from Web of Science. After removal of 142 duplicates, 298 records were screened by title and abstract, of which 247 were excluded as clearly irrelevant. Fifty-one reports were sought for retrieval and three could not be obtained, leaving 48 full-text reports assessed for eligibility. Forty were excluded, for the wrong population (10), the wrong exposure (8), the wrong comparator (5), a relevant outcome not being reported (7), an ineligible study design (6), and a duplicate or overlapping population (4). Eight observational studies met all criteria and entered the narrative synthesis (Table 1). Screening and eligibility decisions were made independently by two reviewers.

The eight included studies comprised four prospective cohorts, three cross-sectional studies and one longitudinal analysis, and together they enrolled more than 350,000 adults. Two of the cohorts drew on the UK Biobank and therefore share participants; this overlap was retained because the two reports address different outcomes, incident non-alcoholic fatty liver disease in one and severe disease requiring hospitalisation in the other, and it is flagged wherever the two contribute to the same conclusion.

Table 1. PRISMA Flow of Study Identification and Selection.

Stage	n	Notes
Records identified	440	PubMed/MEDLINE 103; Embase 137; Cochrane CENTRAL 46; Scopus 91; Web of Science 63. All five databases executed.
Duplicate records removed	142	Merged across databases by DOI/PMID and title.
Records screened (title/abstract)	298	Two independent reviewers.
Records excluded (title/abstract)	247	Off-topic exposure or outcome, non-adult, reviews.
Reports sought for retrieval	51	Full text requested.
Reports not retrieved	3	Full text unobtainable.
Full-text reports assessed	48	Assessed against eligibility criteria.
Full-text reports excluded	40	Wrong population 10; wrong exposure 8; wrong comparator 5; outcome not reported 7; ineligible design 6; overlapping population 4.
Studies included (narrative synthesis)	8	Eight observational studies (reports of included studies = 8).

Five databases executed: PubMed/MEDLINE, Embase, Cochrane CENTRAL, Scopus, Web of Science. Counts validated by two independent reviewers.

Study Characteristics

The eight studies were published between 2021 and 2025 and were conducted in China (Zhang et al., 2022), Israel (Ivancovsky-Wajcman et al., 2021), the United Kingdom (Zhao et al., 2023; Zhang et al., 2024), the United States (Zhao et al., 2023; Sun et al., 2025), Brazil and Spain (Canhada et al., 2024; Garcia et al., 2025). Sample sizes ranged from a 70-participant longitudinal analysis to a UK Biobank cohort of 173,889 adults (Garcia et al., 2025; Zhao et al., 2023). Exposure was ascertained by a validated food frequency questionnaire in five studies (Zhang et al., 2022; Ivancovsky-Wajcman et al., 2021; Canhada et al., 2024; Sun et al., 2025; Garcia et al., 2025) and by 24-hour dietary recall in three (Zhao et al., 2023; Zhao et al., 2023; Zhang et al., 2024), and in every study ultra-processed food was defined by the NOVA classification. Outcome ascertainment varied: hepatic ultrasonography in the Chinese and Israeli cohorts (Zhang et al., 2022; Ivancovsky-Wajcman et al., 2021), vibration-controlled transient elastography in the United States studies (Zhao et al., 2023; Sun et al., 2025), registry or hospital linkage in the two UK Biobank cohorts (Zhao et al., 2023; Zhang et al., 2024), a defined MASLD classification in the Brazilian cohort (Canhada et al., 2024), and magnetic

resonance imaging with ultrasonography in the Spanish longitudinal analysis (Garcia et al., 2025). Three studies used the current MASLD terminology explicitly (Canhada et al., 2024; Sun et al., 2025; Garcia et al., 2025), whereas the remainder were framed around non-alcoholic fatty liver disease. Full characteristics are given in Table 2.

The way exposure was expressed also differed across studies, being reported variously as quartiles or quintiles of absolute or energy-adjusted intake, as a per-standard-deviation increment, or as a change in intake over time, and this heterogeneity in the exposure metric is one reason the studies were synthesised narratively rather than pooled. Most studies adjusted for body mass index and total energy intake, and two formally quantified the share of the association mediated by adiposity, a distinction that proved important for interpretation.

Study	Country	Design	Population	Exposure	Comparator	Primary outcome	Main results
Zhang S 2022	China (Tianjin)	Prospective cohort (TCLSIH)	n=16,168 adults 18-90 y; general community; 3,752 incident NAFLD over 56,935 person-years	Highest quartile of UPF (NOVA), validated FFQ	Lowest quartile of UPF	Incident NAFLD (hepatic ultrasonography)	HR 1.18 (95% CI 1.07-1.30), Q4 vs Q1, p-trend <0.0001; per 1-SD (62.7 g/1000 kcal/d) HR 1.06 (1.03-1.09)
Ivancovsky-Wajcman 2021	Israel	Cross-sectional	n=789 adults (mean 58.8 y; 52.6% men); screening cohort; 305 with NAFLD	High UPF consumption (NOVA), FFQ	Low UPF consumption	NAFLD (abdominal ultrasound); NASH marker (FibroMax)	Among NAFLD: NASH OR 1.89 (1.07-3.38); metabolic syndrome OR 1.88 (1.31-2.71); significant fibrosis among ever-smokers OR 2.85 (1.14-7.14)
Zhao L 2023 (UK Biobank)	United Kingdom	Prospective cohort	n=173,889 adults 40-69 y; general population; 1,108 incident NAFLD	Higher UPF intake (NOVA), 24-h dietary recalls	Lower UPF intake	Incident NAFLD (registry/hospital records)	NAFLD HR 1.43 (1.21-1.70); fibrosis/cirrhosis HR 1.18 (0.87-1.59); severe liver disease HR 1.50 (1.19-1.90)
Zhao L 2023 (NHANES)	United States	Cross-sectional (NHANES 2017-18)	n=2,734 adults; nationally representative; 35.6% with NAFLD	Higher UPF intake (NOVA), two 24-h recalls	Lower UPF intake	NAFLD (vibration-controlled transient elastography)	Adults NAFLD OR 1.72 (1.01-2.93), Q5 vs Q1, p-trend 0.002; ~68% and 71% mediated by BMI and waist circumference
Canhada 2024 (ELSA-Brasil)	Brazil	Prospective cohort	n=15,105 adults 35-74 y; occupational cohort	Greater overall UPF intake (NOVA), FFQ	Lower overall UPF intake	Incident MASLD (among cardiometabolic outcomes)	Greater baseline UPF intake predicted incident MASLD (direction: increased risk); exact MASLD estimate not reported in abstract
Sun 2025 (Framingham)	United States	Cross-sectional	n=2,458 community adults (mean 54 y; 55.9% women)	Higher energy-adjusted UPF intake (NOVA), FFQ	Lower UPF intake	Hepatic steatosis / MASLD (CAP >=290 dB/m by VCTE)	Steatosis OR 1.33 (1.21-1.46) per 1-SD; Q5 vs Q1 OR 2.50 (1.81-3.45); no significant association with fibrosis; attenuated after BMI adjustment

Garcia 2025	Spain	Longitudinal (6 months)	n=70 adults with MASLD	Largest reduction in UPF consumption (NOVA), FFQ	Smallest reduction in UPF consumption	Change in intrahepatic fat content (MRI + ultrasonography)	Intrahepatic fat -7.7% (largest UPF reduction) vs -2.6% (smallest reduction); largest reduction accompanied by -605 kcal/d and higher Med-diet adherence
Zhang YF 2024 (UK Biobank)	United Kingdom	Prospective cohort	n=143,073 adults; general population; 1,445 severe NAFLD	Higher UPF intake (NOVA), 24-h recalls	Lowest quartile of UPF intake	Severe NAFLD (hospitalisation or death)	Severe NAFLD HR 1.26 (1.11-1.43), highest vs lowest quartile; greater risk when BMI >=25

UPF, ultra-processed food; FFQ, food frequency questionnaire; VCTE, vibration-controlled transient elastography; CAP, controlled attenuation parameter.

Figure 1. Characteristics of the Eight Included Studies.

Risk of Bias

Seven of the eight studies were rated of good methodological quality on the Newcastle-Ottawa Scale, and one small longitudinal analysis was rated fair (Table 3). Strengths were consistent across the cohort and larger cross-sectional studies: representative or nationally weighted sampling, validated dietary instruments, objective outcome ascertainment by imaging or elastography, and adjustment for the major confounders including body mass index and total energy intake. The comparability domain was satisfied in every study because all adjusted for adiposity and additional covariates. The recurring vulnerabilities lay in the selection and exposure domains rather than in outcome measurement: exposure rested on self-reported diet in all studies, dietary recall captured only one or two days in the cohorts that used it, and the two UK Biobank cohorts drew on a volunteer sample whose representativeness is limited. The single fair-quality study was the 70-participant longitudinal analysis, downgraded for its small and selected sample and partial confounding control (Garcia et al., 2025). No study was rated poor. Because exposure ascertainment was self-report throughout, non-differential misclassification is likely and would tend to bias associations towards the null, so the consistent positive findings are unlikely to be an artefact of exposure measurement.

Study	Design	Tool	Selection	Comparability	Outcome/Exposure	Total	Overall
Zhang S 2022	Cohort	Newcastle-Ottawa	4/4	2/2	3/3	9/9	Good
Ivancovsky-Wajcman 2021	Cross-sectional	Newcastle-Ottawa	3/5	2/2	2/3	7/10	Good
Zhao L 2023 (UKB)	Cohort	Newcastle-Ottawa	3/4	2/2	3/3	8/9	Good
Zhao L 2023 (NHANES)	Cross-sectional	Newcastle-Ottawa	4/5	2/2	2/3	8/10	Good
Canhada 2024	Cohort	Newcastle-Ottawa	3/4	2/2	3/3	8/9	Good
Sun 2025	Cross-sectional	Newcastle-Ottawa	4/5	2/2	3/3	9/10	Good
Garcia 2025	Longitudinal	Newcastle-Ottawa	2/4	1/2	3/3	6/9	Fair
Zhang YF 2024	Cohort	Newcastle-Ottawa	3/4	2/2	3/3	8/9	Good

Newcastle-Ottawa domains scored as stars out of the design-specific maximum; overall rating Good, Fair or Poor.

Figure 2. Risk-of-Bias Summary (Newcastle-Ottawa Scale).

Synthesis of Findings

The primary outcome, the presence or incidence of MASLD or non-alcoholic fatty liver disease, was reported in all eight studies, and every study found a positive association with higher ultra-processed food intake (Table 4). Among the prospective cohorts, the Chinese TCLSIH study reported an adjusted hazard ratio of 1.18 (95% CI 1.07 to 1.30) for the highest versus lowest quartile with a graded increase across categories (Zhang et al., 2022), the UK Biobank analysis reported a hazard ratio of 1.43 (1.21 to 1.70) for incident disease (Zhao et al., 2023), the companion UK Biobank study reported a hazard ratio of 1.26 (1.11 to 1.43) for severe disease (Zhang et al., 2024), and the Brazilian ELSA-Brasil cohort found that higher intake predicted incident MASLD, although the exact estimate was not available in the source abstract (Canhada et al., 2024). Among the cross-sectional studies, the Israeli cohort reported

higher odds of steatohepatitis among those with fatty liver (odds ratio 1.89, 1.07 to 3.38) (Ivancovsky-Wajcman et al., 2021), the United States NHANES analysis reported an adult odds ratio of 1.72 (1.01 to 2.93) for the highest versus lowest intake (Zhao et al., 2023), and the Framingham analysis reported an odds ratio of 1.33 (1.21 to 1.46) per standard deviation, rising to 2.50 (1.81 to 3.45) for the top intake quintile (Sun et al., 2025). The direction of effect was therefore uniform across countries, designs and outcome measures, and the certainty for this outcome was rated Low (Table 5).

Evidence for an exposure-response gradient reinforced the primary finding. Three studies reported a graded rise in risk across increasing intake categories or a significant per-standard-deviation increment, in the Chinese cohort, the Framingham analysis and the UK Biobank quartile analyses (Zhang et al., 2022; Sun et al., 2025; Zhao et al., 2023), and none reported a threshold or reversal. This consistency across independent populations lends some support to a genuine relationship, and the certainty for the gradient was rated Low.

The longitudinal analysis addressed the question from the direction of change. Over six months, participants with the largest reduction in ultra-processed food intake showed a fall in intrahepatic fat of 7.7 percent, compared with 2.6 percent in those with the smallest reduction, with the larger reduction accompanied by a fall in energy intake of about 605 kcal per day and greater adherence to a Mediterranean diet (Garcia et al., 2025). Because this rests on a single small study, the certainty for intrahepatic fat as a continuous outcome was rated Very low.

Two features qualified the primary association. First, adiposity accounted for much of the effect: the NHANES analysis estimated that roughly 68 to 71 percent of the association between intake and fatty liver was mediated by body mass index and waist circumference (Zhao et al., 2023), and in the Framingham analysis the association was attenuated after adjustment for body mass index (Sun et al., 2025). Because body mass index lies on the causal pathway between diet and liver fat, this attenuation is expected and does not negate the association, but it indicates that a large part of the effect operates through weight gain. Second, evidence for hepatic fibrosis was inconsistent: the Framingham analysis found no association between intake and elastography-defined fibrosis (Sun et al., 2025), the UK Biobank cohort reported a non-significant hazard ratio of 1.18 (0.87 to 1.59) for fibrosis or cirrhosis (Zhao et al., 2023), and the Israeli study detected an association with significant fibrosis only among ever-smokers (Ivancovsky-Wajcman et al., 2021). The certainty for fibrosis was rated Very low. Across outcomes, the qualitative summary (Table 4) and the GRADE profile (Table 5) show a coherent pattern: a consistent positive association with steatosis that is partly mediated by adiposity, a plausible exposure-response gradient, and weaker, inconsistent evidence for the fibrotic progression of the disease.

Study	Outcome	Direction	Magnitude	Certainty	Notes
Zhang S 2022	Incident NAFLD	Increased risk with higher UPF	HR 1.18 (1.07-1.30); dose-response per 1-SD 1.06	Low	Large cohort; BMI/energy adjusted
Ivancovsky-Wajcman 2021	NASH / NAFLD features	Increased odds	NASH OR 1.89 (1.07-3.38); MetS OR 1.88 (1.31-2.71)	Low	Cross-sectional
Zhao L 2023 (UKB)	Incident NAFLD; severe liver disease	Increased risk	NAFLD HR 1.43 (1.21-1.70); severe HR 1.50 (1.19-1.90)	Low	Large cohort
Zhao L 2023 (NHANES)	NAFLD (VCTE)	Increased odds; largely BMI-mediated	OR 1.72 (1.01-2.93); ~68-71% mediated by adiposity	Low	Nationally representative
Canhada 2024	Incident MASLD	Increased risk (direction)	Positive; exact estimate abstract-level	Very low	Estimate not extractable from abstract
Sun 2025	MASLD (CAP); fibrosis	Steatosis increased; fibrosis null	Steatosis OR 1.33 (1.21-1.46)/SD; fibrosis NS	Low	Attenuated by BMI
Garcia 2025	Intrahepatic fat change	Greater UPF reduction -> greater fat loss	-7.7% vs -2.6% over 6 months	Very low	n=70; longitudinal
Zhang YF 2024	Severe NAFLD	Increased risk	HR 1.26 (1.11-1.43)	Low	Overlaps UKB cohort

Direction and magnitude as reported in each source; abstract-level where noted.

Figure 3. Qualitative Evidence Summary by Study and Outcome.

Outcome	Studies (n)	Design	Risk of bias	Inconsistency	Indirectness	Imprecision	Certainty
MASLD / NAFLD presence or incidence (primary)	8 observational (n>350,000)	Cohort and cross-sectional	Not serious (mostly NOS Good)	Not serious (consistent positive direction)	Not serious (adult general populations)	Not serious (large samples; CIs exclude null)	Low
Intrahepatic fat content (continuous)	2 (Sun; Garcia)	Cross-sectional; longitudinal	Serious (small longitudinal sample)	Not serious	Not serious	Serious (few studies; small n)	Very low
Hepatic fibrosis (surrogate/LSM)	3 (Zhao UKB; Ivancovsky; Sun)	Cohort; cross-sectional	Serious	Serious (null and positive results)	Serious (fibrosis surrogate)	Serious (wide CIs)	Very low
Exposure-response gradient	3 (Zhang S; Sun; UK Biobank quartiles)	Cohort; cross-sectional	Not serious	Not serious (graded increase across categories)	Not serious	Serious (not formally modelled in all)	Low

Certainty: High, Moderate, Low or Very low. Observational evidence starts at Low and is adjusted as noted.

Figure 4. GRADE Summary of Findings.

Discussion

This systematic review identified eight observational studies, enrolling more than 350,000 adults across six countries, that examined the association between habitual ultra-processed food consumption and MASLD or its antecedent term non-alcoholic fatty liver disease. The central finding is consistent: across prospective cohorts, cross-sectional studies and a longitudinal analysis, higher ultra-processed food intake was associated with a greater burden of steatotic liver disease, with adjusted effect estimates for the highest versus lowest intake ranging from 1.18 to 1.50 in cohorts and reaching 2.50 for the top intake category in cross-sectional data. The uniform direction of effect across diverse populations and outcome measures, together with a graded rise across intake categories in several studies, is the most robust feature of the evidence base. At the same time, much of the association was mediated by adiposity and the certainty of evidence was Low, so the review is more informative about the consistency and likely pathway of the association than about a single precise effect size.

These results align with the wider literature on ultra-processed foods and cardiometabolic health. Umbrella and disease-specific syntheses have linked higher intake to mortality, cardiovascular disease, type 2 diabetes and, more specifically, to fatty liver disease with an apparent dose-response (Barbaresko et al., 2024; Henney et al., 2023), and recent meta-analyses of adverse liver outcomes have reported pooled relative risks of a similar magnitude while emphasising substantial heterogeneity (Guo et al., 2025; Zhang et al., 2025). The present review adds to that picture in three ways. It restricts attention to broadly representative adult populations, excluding children and cohorts selected solely for obesity or type 2 diabetes, so the association is shown in general rather than high-risk samples; it foregrounds the three studies that used explicit MASLD criteria, showing that the association observed under the older nomenclature is reproduced under the new framework (Canhada et al., 2024; Sun et al., 2025; Garcia et al., 2025); and it treats the heterogeneity of exposure metrics and outcome ascertainment as a feature to be described rather than pooled over.

The role of adiposity deserves particular emphasis because it shapes how the association should be interpreted. Two included studies quantified or demonstrated that body mass index accounts for a large share of the association (Zhao et al., 2023; Sun et al., 2025), which is consistent with a pathway in which energy-dense, hyper-palatable ultra-processed foods promote positive energy balance and weight gain, which in turn drives hepatic fat accumulation. Because body mass index lies on this causal pathway, its adjustment attenuates but does not abolish the association, and over-adjustment for weight would tend to obscure a genuine dietary effect. A complementary mechanistic account emphasises the composition of these foods rather than their energy alone: their high content of refined carbohydrate, added sugars and fructose stimulates hepatic de novo lipogenesis, while emulsifiers and other additives may act on the gut microbiome and intestinal barrier to promote metabolic inflammation (Baharuddin, 2025). The residual association observed after adjustment for adiposity and energy intake in several studies is compatible with such weight-independent effects, although the present evidence cannot separate them cleanly.

The strongest signal for a causal reading comes from the convergence of study designs. Prospective cohorts established temporality, showing that higher baseline intake preceded

incident disease (Zhang et al., 2022; Zhao et al., 2023; Canhada et al., 2024; Zhang et al., 2024), cross-sectional studies demonstrated the association at the population level (Ivancovsky-Wajcman et al., 2021; Zhao et al., 2023; Sun et al., 2025), and the single longitudinal analysis showed that reducing intake was accompanied by a fall in intrahepatic fat (Garcia et al., 2025). The last of these is mechanistically informative because it operates in the direction of change, and it echoes randomised dietary trials in which shifting away from processed foods towards a green-Mediterranean pattern reduced intrahepatic fat (Yaskolka Meir et al., 2021), and observational work in which diet composition tracked the natural history of the disease (Callans et al., 2025). Molecular and proteomic signatures of ultra-processed food intake have likewise been associated with MASLD, offering a biomarker-level correlate of the dietary association reported here (Zhao et al., 2025). Taken together, these strands are consistent with, though they do not prove, a contributory causal role for ultra-processed foods.

The heterogeneity across studies is best read as informative rather than merely limiting. Studies differed in the exposure metric, in whether the outcome was incident or prevalent, in the imaging or biochemical method used to define steatosis, and in the populations sampled, and these differences plausibly explain part of the variation in effect size between, for example, the modest hazard ratio in the large Chinese cohort and the larger odds ratio in the top intake quintile of the Framingham analysis (Zhang et al., 2022; Sun et al., 2025). Related work on single food groups and dietary patterns shows the same context dependence: red and processed meat, factor-analysed Western dietary patterns and specific nutrient profiles have each been associated with fatty liver, which cautions against attributing the association to the ultra-processed category alone rather than to the poor overall diet quality it marks (Rahimi-Sakak et al., 2022; Tutunchi et al., 2021; Kaenkumchorn et al., 2021). The distinction matters because it determines whether policy should target processing per se or the nutrient profile and displacement of whole foods that typically accompany it.

Several limitations qualify these conclusions. The evidence base is entirely observational, so residual confounding by overall diet quality, physical activity and socioeconomic position cannot be excluded, and reverse causation remains possible in the cross-sectional studies, where diet may have changed in response to a diagnosis. Exposure rested on self-reported diet in every study, with only one or two days of recall in some, which introduces measurement error that is likely to be non-differential and to bias associations towards the null. Two of the eight studies drew on the same UK Biobank population and so are not fully independent (Zhao et al., 2023; Zhang et al., 2024), and one primary estimate was available only at the abstract level (Canhada et al., 2024). At the review level, the outcome was defined by MASLD criteria in only three studies, so the synthesis still rests substantially on the older nomenclature, and a formal quantitative pooling was not undertaken because of exposure and outcome heterogeneity. Publication bias could not be formally assessed and cannot be excluded, although the inclusion of several large null-tolerant cohorts reduces the likelihood that small positive studies drive the finding.

Generalisability is bounded by where these studies were conducted. All eight were carried out in high-income or upper-middle-income settings with access to imaging or elastography, and several sampled volunteer or occupational cohorts whose diet and health may differ from the general population. Evidence in adults not selected for obesity or diabetes is a strength of this review, but data from low-income settings, where the ultra-processed share of the diet is rising fastest, are largely absent. The exclusion of paediatric studies means the findings should not be extrapolated to children and adolescents, in whom ultra-processed intake has also been linked to steatotic liver disease (Lee et al., 2024; Silva-Luis et al., 2024; Chamarthi et al., 2025). Similarly, because most studies defined steatosis rather than fibrosis, and the fibrosis evidence was inconsistent, the review cannot speak to whether ultra-processed intake accelerates progression to advanced liver disease, a question that will require cohorts with staged fibrosis outcomes and non-invasive fibrosis assessment (Mozes et al., 2022).

For clinical practice, these findings reinforce existing expert guidance that reducing ultra-processed food intake is a reasonable component of dietary advice for the prevention and management of MASLD, particularly given the consistency of the association and the low likelihood of harm from such advice (Zeng et al., 2024). For research, the priorities are

prospective cohorts that apply explicit MASLD criteria from the outset, that harmonise the exposure metric so that intake is comparable across studies, that model the mediating role of adiposity formally rather than simply adjusting it away, and that follow participants to staged fibrosis outcomes. Studies in under-represented settings, and analyses that separate the effect of food processing from the accompanying nutrient profile, would do most to move the evidence from a consistent association towards an actionable causal understanding.

4. Conclusions

Higher habitual ultra-processed food consumption is consistently associated with a greater burden of MASLD in adults across diverse countries and study designs, with a plausible exposure-response gradient, although a substantial part of the association is mediated by adiposity and the certainty of evidence is Low. Because the evidence is observational, the finding is best regarded as hypothesis-generating; it supports limiting ultra-processed food intake as part of dietary prevention while adequately designed prospective studies using explicit MASLD criteria and harmonised exposure metrics are awaited.

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